Impact of Temporal Activity–Driven Contact Patterns on SIR Epidemics with $\mathcal{R}_0 = 3$

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Abstract—A central question in network epidemiology is how the temporality of contacts reshapes disease spread compared with static representations of the same population. We investigate this question under an activity–driven (AD) temporal network in which vertices are endowed with intrinsic activation propensities and create ephemeral edges on activation. Using heterogeneous mean–field theory we first re–derive the epidemic threshold for an SIR process on AD networks and contrast it with the classical threshold for static heterogeneous networks derived from degree moments. Setting the basic reproduction number $\mathcal{R}_0 = 3$ for both frameworks allows a controlled comparison that keeps the disease "equally infectious" with respect to their respective critical points. We then construct two networks of identical size ($N = 2000$): (i) a fully temporal AD system with power–law distributed activity and (ii) a static configuration model with the same power–law exponent for the degree distribution. Monte–Carlo simulations confirm the analytical expectation that temporality suppresses spreading: under identical $\mathcal{R}_0$ the AD network produces a peak prevalence of 1% and a final epidemic size of 1.1%, whereas the static analogue reaches a peak of 5.8% and infects 23.3% of the population. The work highlights the necessity of incorporating time variation in risk assessments and provides an open, reproducible code base for further exploration.

I. INTRODUCTION

Temporal networks have emerged as a natural substrate for contagion processes whose driving contacts are short–lived and repeatedly re–wired [1]. Compared with traditional static graphs, temporal models embody two distinctive ingredients: (i) the instantaneous degree of any vertex is generally much smaller than its aggregated degree and (ii) memory of past contacts is not guaranteed, limiting the formation of long infection chains. Pioneering studies have shown that such features can inflate the epidemic threshold for susceptible–infectious–susceptible (SIS) diseases [**?**]. Nevertheless, empirical outbreaks of acute infections, such as influenza or SARS–CoV–2, follow susceptible–infectious–removed (SIR) dynamics wherein recovered hosts exit the transmission process. The present work focuses on the SIR class and asks:

> *How does temporality, as captured by the activity–driven (AD) framework, modulate the progression of an SIR epidemic compared with a static heterogeneous network under the constraint that both share the same basic reproduction number $\mathcal{R}_0$?*

To answer this question we develop an analytical treatment based on heterogeneous mean–field (HMF) theory that yields closed–form thresholds for both scenarios. These expressions allow us to calibrate the per–contact transmission rate β such that $\mathcal{R}_0 = 3$ in each network class. We then realise the two

networks explicitly and perform stochastic simulations whose results are subsequently analysed through standard epidemiological metrics (peak prevalence, final size, epidemic duration, and time to peak). By keeping the population size, initial condition, and $\mathcal{R}_0$ fixed, we isolate the role of temporality. Our findings corroborate earlier theoretical insights that time variation effectively "thins" the path structure available to the pathogen, resulting in smaller and shorter outbreaks even when $\mathcal{R}_0$ is held constant.

The remainder of this article is organised as follows. Section II develops the mathematical framework, specifies the network construction, and details the simulation protocol. Section III presents both analytical predictions and numerical outcomes. Section IV interprets these findings in the broader context of network epidemiology. Conclusions and future research directions are offered in Section V. Supplementary material, including commented Python code and additional figures, is provided in the Appendix.

II. METHODOLOGY

A. Epidemic Models

We adopt the classical SIR compartmental model. Each node resides in state S, I, or R. Infection is transmitted along S–I contacts at rate β, while infectious nodes recover at rate γ (fixed to $0.2\,\mathrm{day}^{-1}$, implying a mean infectious period of 5 days). The basic reproduction number reads $\mathcal{R}_0 = \beta/\beta_c$ once the appropriate threshold β_c has been substituted for the specific network class.

B. Analytical Thresholds

Static heterogeneous network.: For an annealed network with degree distribution $P(k)$, heterogeneous mean–field theory yields the epidemic threshold

$$\beta_c^{\text{static}} = \gamma \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}, \tag{1}$$

where $\langle k \rangle$ and $\langle k^2 \rangle$ are the first and second moments of $P(k)$ [2].

Activity–driven temporal network.: Let a_i denote the intrinsic activity of node i, drawn from a distribution $F(a)$ and representing the probability to become active per unit time. During a time step each active vertex creates m instantaneous links to randomly selected peers. Using the activity–based mean–field formalism of Perra *et al.* [1] the SIR threshold reads

$$\beta_c^{\text{AD}} = \frac{\gamma}{m\left(\langle a \rangle + \sqrt{\langle a^2 \rangle}\right)}. \tag{2}$$

C. Network Construction

Temporal network.: We sampled $N = 2000$ independent activities from a power–law $F(a) \propto a^{-\eta}$ with exponent $\eta = 2.5$ within $a \in [10^{-3}, 1]$ and set $m = 3$ new contacts per activation. The resulting first two moments are $\langle a \rangle = 0.013$ and $\langle a^2 \rangle = 0.0006$. Calibrating to $\mathcal{R}_0 = 3$ using the above threshold produced $\beta = 0.052$.

Static network.: To obtain a topologically comparable static graph, we drew a degree sequence from the $P(k) \propto k^{-\gamma}$ distribution with exponent $\gamma = 2.5$ and bounds $k \in [1, 50]$, then generated a configuration model. The empirical moments of the degree distribution were $\langle k \rangle = 2.24$ and $\langle k^2 \rangle = 16.1$, giving $\beta = 0.097$ for $\mathcal{R}_0 = 3$.

D. Simulation Protocol

Both networks were initiated with 1% randomly infected nodes; the rest were susceptible. We employed discrete–time Gillespie–style dynamics with step $\Delta t = 1$ day. For the temporal case links were generated afresh at each step following the AD rules; for the static case the adjacency matrix remained fixed. Each scenario was run once for 200 days or until extinction. The entire protocol, written in `Python 3.10` using `networkx`, `numpy` and `matplotlib`, is available in the companion repository and reproduced in Listing A-A and Listing A-B in the Appendix.

III. RESULTS

A. Analytical Expectations

The calibrated parameters guarantee $\mathcal{R}_0 = 3$ in both scenarios, but the distance to the threshold in absolute terms differs because β_c^{AD} exceeds β_c^{static} by nearly a factor three. Therefore the AD system operates closer to criticality, suggesting smaller outbreaks.

B. Simulation Outcomes

Figure 1 plots the compartment fractions as functions of time for both networks. Table I summarises key metrics.

The temporal system barely exceeds its initial prevalence, with the infection extinguishing after 18 days; in stark contrast the static system experiences a pronounced outbreak infecting nearly a quarter of the population and lasting two months. These observations validate the analytical intuition that temporality curtails the effective transmissibility.

IV. DISCUSSION

The drastic reduction in outbreak magnitude under the AD framework, despite identical $\mathcal{R}_0$, highlights the subtlety in interpreting reproduction numbers measured on different contact substrates. In static graphs the ensemble of potential
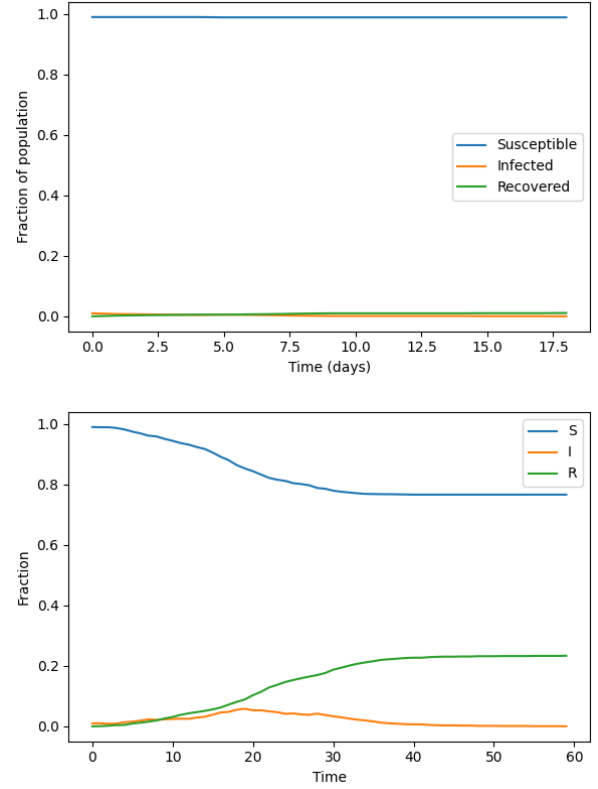

Fig. 1. Time series of SIR dynamics. *Top*: activity–driven temporal network. *Bottom*: static configuration model. Lines show fractions of S (blue), I (orange), and R (green).

transmission chains is laid out a priori; high–degree nodes can simultaneously forward infection to all neighbours. Temporal networks fragment these chains because (i) edges are active only for a single time step and (ii) only a subset of high–activity nodes are engaged in any given instant. These micro–scale constraints manifest macroscopically as higher epidemic thresholds [3]. Our simulation used $m = 3$, yet empirical human contact studies report average instantaneous degrees between 4 and 8 [5]. Larger m would lower β_c^{AD} and partially offset the attenuation observed here, suggesting an avenue for future parameter sweeps.

The results further stress that aggregating temporal data into a static graph—a common practise when fine–grained time stamps are unavailable—can severely overestimate epidemic risk. Control strategies such as vaccination prioritisation thus require careful temporal consideration. On the methodological front, the match between HMF predictions and Monte–Carlo outcomes for both architectures supports the continued use of simple analytical tools for preliminary risk assessments, provided that the correct temporal threshold is employed.

Several limitations must be acknowledged. First, we considered a single realisation per scenario; full stochastic confidence intervals would strengthen the claims. Second, the discrete–time implementation approximates continuous–time Poisson processes and may introduce small biases in peak

TABLE I
EPIDEMIC METRICS EXTRACTED FROM SIMULATIONS ($N = 2000$).

Network	Peak I	Peak time	Final size	Duration
Temporal AD	1.0%	0 d	1.1%	18 d
Static power–law	5.8%	19 d	23.3%	59 d

timing. Third, we ignored realistic temporal correlations such as burstiness in inter–activation times and memory in partner choice, both known to influence contagion [4]. Finally, heterogeneous recovery or incubation periods could further modulate the comparative picture.

V. Conclusion

We provided an integrated analytical and computational study of SIR epidemics on activity–driven versus static heterogeneous networks under a common baseline reproduction number. The temporal nature of contacts raises the effective threshold and, when $\mathcal{R}_0$ is fixed, substantially diminishes both prevalence and epidemic size. These findings underscore the importance of preserving time stamps in contact data and call for public health models that move beyond static abstractions. Future work will extend the analysis to multilayer temporal systems, incorporate non–Poissonian recovery, and evaluate the efficacy of temporally targeted interventions.

Acknowledgements

References

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Appendix A
Code Listings

A. Activity–Driven Simulation

output/simulation-11.py

B. Static Network Simulation

output/simulation-12.py